

Subject 3's records show no evidence of reacquisition although performance measured in number of correct responses does show an increase in R1 and R2 over B1, B2, S1, and S2. Subjects 3 and 5 demonstrate increased variability in acquisition and reacquisition.

There was no consistent response to the question, "What did you do to get rewarded?" The responses were of three types: (i) imagined sights such as "I imagined seeing a pin stick me in the head each time I heard the tone"; (ii) imagined sounds such as, "I heard a [second] tone"; and (iii) special attention to various aspects of the stimulus, for example, "I tried not to hear just one tone but an *on* part and an *off* part." Of the 12 subjects in experiments 1 and 2, four responses were of type (i), four were of type (ii), and two were of type (iii), with two subjects reporting that they had to change their strategy from time to time, sometimes imagining a sound, sometimes a sight, with a variety of specific imagery.

The operant control demonstrated here is far from large. These subjects at best did not exceed 30 percent successful responses when chance success was about 16 percent.

It should be noted that the averaged amplitude increases in *acquisition* and *reacquisition* are not apparent although the increases in success scores (Fig. 1) are. Such results agree with the small absolute size of the effect of reinforcement and may be explained if one assumes that, during acquisition trials, a subject may show an increase in criterion responses while missing criterion on unsuccessful trials by a wider margin than during unsuccessful trials of *base* and *suppress*.

Our data do not offer any simple explanation of the operant control phenomenon. Clearly, subjects are not able to quickly perfect the response. Some subjects volunteered the information that immediate feedback on the oscilloscope in experiment 1 was more a hindrance than a help. They said it distracted them from the intense concentration that they needed to do well in the task. If some simple behavior regularly resulted in the rewarded neural event, subjects watching the oscilloscope should have been more quickly able to discover the right technique. There seems, however, to be little difference in the scores of the two experiments. The variety of verbal reports and the various types of changes seen in the average evoked potentials

in the criterion segment and elsewhere argue against the idea that subjects can learn a simple motor response whose somesthetic feedback or efferent command generates the rewarded amplitude change. In view of the controls for systematic movements, such an interpretation becomes even less tenable. The use of earphones as the vehicle of stimulus presentation makes it unlikely that learned changes in receptor orientation are the simple explanation of the phenomena. This general kind of interpretation might, however, be successfully revised to account for the phenomenon by basing it upon the notion that subjects can learn to attend (or not to attend) to the stimulus, behavior whose neural correlate could be an enhanced component (5). Yet Fox and Rudell reported two successfully conditioned voltage changes of opposite direction. It seems unlikely that control of attention could be mastered with such specificity.

The lack of uniformity in verbal and neural responses makes it difficult to propose a specific mechanism for the operant control reported, even if we have eliminated notions involving a regularly occurring neural correlate (efferent or afferent) of a movement. It is likely that subjects are learning to generate some internal state which

may mediate an altered evoked potential by either increasing the overall excitability of many neuron populations, or by increasing the size of a particular population so that when the population is activated, its greater effective size yields an enhanced voltage (6). The verbal reports of the subjects suggest that behavior they call "imagining" can bring the relevant state about.

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  7. Supported by PHS grant MH11834 to S.S.F.
- 6 February 1969; revised 5 May 1969 ■

## Pulmonary Gas Transplant Time

The experimental evidence presented by Wagner *et al.* (1) does not imply that there is an interaction between the bulk flow and diffusion. On the contrary, the C-shaped curve in Fig. 1 suggests that the mechanism of transport is by convection alone [a more detailed discussion of convective dispersion is given in (2)]. The fact that the shortest transit time is about half that obtained from a calculation based upon the average velocity would also result if the velocity profile in the ducts are nearly parabolic; when the Reynolds number of the flow is less than about 2100, the velocity profile is nearly parabolic and the maximum velocity within the duct is nearly twice the average velocity. Furthermore, because Taylor diffusion (3) involves only radial diffusion, the minimum transit time does not change; that is, the minimum transit time remains equal to the distance between the larynx and the alveolus, divided by the velocity of the fastest streamline.

This is not to say that Taylor diffusion is not occurring. Ross (4) has indicated the gross structure of the dog's bronchial tree. However, to determine whether or not Taylor diffusion is important, the characteristic time associated with convective dispersion must be compared with that for the decay of radial variations in concentrations. Taylor diffusion would be expected to occur in the small diameter bronchi.

However, before the question of transport mechanism can be resolved, the characteristic times must be compared for each branch in the bronchial tree followed by the appropriate dispersion analysis; then the results should be carefully compared with the shape of the residence time distribution curve associated with a step change in stream concentration.

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28 March 1969

It is difficult to visualize convective dispersion, accounting for the short pulmonary gas transport times we measured; convective dispersion requires well-developed parabolic velocity profiles where the central streamlines move at twice the average velocity. Laminar profiles of this kind are unlikely to develop within the rapidly arborizing bronchial tree (gas molecules arriving at an average alveolus must traverse  $\cong 20$  generations of branches with a distance of only 3.5 diameters between bifurcations) (*1*). In this system of branching tubes, the faster-moving molecules in the center of one bronchus are divided at the branching point so that they tend to be moved into the slow-moving streamlines near the walls of the next branches, thus keeping convective dispersion to a minimum. Rapid radial diffusion in these narrow tubes further minimizes radial gas concentration gradients making significant convective dispersion even more improbable.

Because the bronchial tree is not open-ended, a tidal volume of 360 ml cannot be expected to reach the alveolar surface in a lung with a volume of 3600 ml by convective dispersion alone or, for that matter, by any other form of bulk flow. Indeed it can be shown by conservative calculations that diffusion is the primary mechanism of transport for the terminal 2 cm of the airways. According to Cumming *et al.* (*2*), still gas diffusion alone would require on the order of 1 second to transport detectable quantities of carbon monoxide over this distance; so even if we assume 0 time for bulk flow, still gas diffusion alone would require more time than we measure for the entire process. Obviously, diffusion must be facilitated in some way. In applying the Taylor analysis (*3, 4*) to the lung, radial diffusion reduces radial concentration gradients very rapidly. There does remain, however, a significant longitudinal concentration gradient which is the basis of our speculation concerning bulk flow-diffusion interaction. We agree, as we stated in our report, that the characteristic times must be compared for each branch in

the bronchial tree before the question of transport mechanisms can be resolved. We also agree that the shape of the curve of carboxyhemoglobin formation requires further investigation.

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5 June 1969

## Statistics of Unseen Animals

Hanson (*1*) has proposed a new method for estimating the abundance of animal populations based on successive surveys. The ecologist is often confronted with the problem that the organisms are not all "seeable" or "catchable" by the methods available to him; this results in a sample which constitutes an unknown proportion of the total population. Hanson's procedure seems, in some remarkable way, to overcome this problem. Unfortunately, however, this conclusion is mistaken because of fundamental errors in the derivation.

It is proposed in Hanson's Eq. 4 that

$$y_1 = Y_1 (x_1/K)$$

where  $y_1$  is the number of unseen animals occupying spaces in which one or more animals were actually seen,  $Y_1$  is the total number of unseen animals,  $x_1$  is the number of observed animals, and  $K$  is the population size. Since  $Y_1$  is always less than  $K$ , this equation has the interesting but unrealistic property that  $y_1$  is always estimated to be less than  $x_1$ .

Consider the case in which 1000 animals are present, on a grid of 100 spaces, with  $P$ , the probability that any given animal would be seen, equal to .10. One would expect, in a complete survey, to see approximately 100 animals and, since

$$Y_1 = K - x_1$$

or 900, Hanson's Eq. 4 would propose that there are 90 animals unseen on those grid spaces on which these 100 animals were seen. If we may assume,

as Hanson suggests, that  $P$  is unaffected by distribution, then (i) if the animals are extremely patchy, say, all on one grid space,  $y_1$  would in fact be 900 rather than 90; (ii) if the animals are randomly distributed, one should expect to see these 100 animals on a total of about 63 spaces (Poisson distribution), which would actually contain about 630 animals, of which 100 were seen; this would lead to an expected true value for  $y_1$  of about 530 instead of 90; (iii) if the animals were evenly distributed, ten per space, one should expect to see the 100 animals on a total of about 65 spaces (binomial expansion), and  $y_1$  would have a true value of about 550; and (iv) in the unlikely case that all 100 animals were seen one per space (biased observation methods),  $y_1$  would be 900. Clearly, regardless of distribution, Eq. 4 gives extremely bad estimates of  $y_1$  in this example; and, in fact, after much manipulation of such hypothetical cases, we are convinced that it is a very rare situation in which the estimates obtained are at all reasonable.

The quantity  $Y_1 (x_1/K)$  should, under Hanson's assumptions, be an unbiased estimator of the number of animals to be seen in a second, complete census, provided that the animals seen in the first census ( $x_1$ ) were in some manner removed from further consideration. There appears to be no defensible logic, however, that would lead one to expect that this quantity will bear any consistent relationship to  $y_1$ , the parameter which it is purported to estimate; the above example indicates the kind of inconsistency to be encountered.

If Eq. 4 is inappropriate for estimating  $y_1$ , then Hanson's Eq. 5 is also in error, since it is derived from Eq. 4. In the four cases considered in the above example, Eq. 5 would lead to estimates of total population size of 100, about 250, about 250, and 100, respectively, rather than the known value of 1000. Since Eq. 5 is not an appropriate estimator for  $K$ , it follows that Hanson's Eq. 6 is also in error, that is,  $(x_2/x_1)^{1/2}$  is not identical with  $(1 - P)$ , nor is it a reasonable estimator of that parameter.

Hanson's Eq. 7 purports to provide an estimator of  $y_1$  when the distribution is even:

$$y_1 = Y_1 (l_1/L_1)$$

where  $l_1$  is the number of spaces on which animals were seen, and  $L_1$  is the